Modern Concepts of Cardiovascular Disease

Published monthly by the AMERICAN HEART ASSOCIATION
450 SEVENTH AVENUE, NEW YORK, N. Y.

DR. SAMUEL A. LEVINE, Boston, *Editor*
DR. MARSHALL N. FULTON, Boston, *Associate Editor*

Vol. II **March, 1933** **No. 3**

HEART DISEASE AND ITS RELATION TO TUBERCULOSIS

Symptoms of circulatory imbalance during the course of pulmonary tuberculosis are legion. In some instances they are the evidences of associated cardiovascular disease, whereas in others they are among the manifestations of altered function associated with infection. In the main, it is true that tuberculosis neither predisposes to nor insures against organic disease of the heart and it may be that the relative infrequency of myocardial failure even if there is a structural defect of the heart, is determined partly by the beneficent influence of the restful regimen of life that is instituted.

In given cases, certain evidences of altered circulatory activity may raise doubts as to the anatomical status of the heart and the vessels and it is essential that the significance of such be realized if diagnostic and therapeutic pitfalls are to be avoided. Some of the more usual of these merit particular consideration.

Neurocirculatory asthenia, vasomotor instability, effort syndrome, designate as one will, the grouping of palpitation, tachycardia, lability of the pulse, transitory erythemas, sweats, breathlessness, sensations of substernal oppression, irritability and other ill-defined somatic discomforts, are encountered oftentimes in individuals with loci of pulmonary tuberculosis. Failure to regard this syndrome as symptomatic of the infection may lead to the diagnosis of thyrotoxicosis or of primary circulatory disease. Conviction that there is actual disease of the heart may seem to be warranted by the detection in the second left interspace near the sternum of a visible and palpable pulsation, an accentuated pulmonic second sound and a systolic murmur. To the informed, these evidences of an uncovered conus arteriosus are too well known to require more than mention. Intensification or reduplication of the second cardiac sound in the pulmonic area if associated with a systolic murmur in the mitral area may arouse suspicion of a mitral insufficiency but unless the first sound is sharpened or a late diastolic murmur is heard, organic disease of the mitral ring need not be considered seriously. Conflicting views are held as to the influence of mitral stenosis upon the development of phthisis but certain it is that the latter occurs only rarely if there is a marked narrowing of the mitral orifice and mitral stenosis occurs seldom in pulmonary tuberculosis. Experience indicates no valid reason for a protective or for a predisposing influence of the one state upon the other and suggests that whatever apparent antagonism there may be, is a chance one. On the other hand, pulmonary tuberculosis is a usual development in congenital stenosis of the pulmonary orifice. The coexistence of rheumatic or of syphilitic disease of the heart and phthisis though encountered now and again is to be considered an accidental association of importance chiefly as a determinant of the modification of therapeutic procedures.

Study of individuals of the so-called asthenic or ptotic habitus has demonstrated that there is no fundamental correlation between a so-called vertical or dropped heart and tuberculosis of the lungs nor between hypoplasia of the heart and great vessels and the latter disease. The vertical position and the small size of the cardiovascular X-ray shadow are usual findings in long standing tuberculosis and are perhaps to be interpreted as the result of bodily configuration intensified by physical inactivity and wasting. Functional murmurs are to be heard in many tuberculous patients and of these a cardiorespiratory-inspiratory or a blowing systolic bruit at the apex, over the conus or over the precordium are the most usual. The absence of associated modification of the valvular sounds and of cardiac enlargement suffice generally to identify them.

Tuberculous endocarditis is a rare disease and is seldom recognized clinically. It may give rise to valvular or to mural lesions, the bacteriogenesis of which may perhaps be suspected only if these

evidences develop in the course of a generalized tuberculosis uncomplicated by a secondary infection.

Pericarditis due to the tubercle bacillus is a strangely infrequent complication of phthisis despite the evident opportunity for invasion of the pericardium by contiguity or by metastasis. On the contrary, tuberculous pericarditis develops often as one localization of tubercles in polyserositis, as a consequence of mediastinal or spinal tuberculosis or as the sole clinical manifestation of tuberculosis. Serofibrinous as a rule, it may be seropurulent, serosanguinous or it may be of the plastic, obliterative type from the onset. It is an interesting fact that in polyserositis there are seldom any demonstrable active pulmonary loci of tuberculosis. Calcification of the pericardium may follow upon an acute or a subacute pericarditis or it may occur in individuals from whom no history can be elicited to suggest an antecedent pericardial infection.

Dyspnea and tachycardia are usual symptoms even in the earliest recognizable stages of pulmonary tuberculosis; speeding of the cardiac rate and the occurrence of extra systoles are apparent often after minimum stimuli and syanosis and edema are seen in many cases of advanced phthisis. The complaint of palpitation or of precordial distress with or without exertion is likewise heard frequently. An actual myocardial failure is not encountered with any appreciable regularity in tuberculosis of the lungs. Livid cyanosis, dyspnea and enlargement of the right side of the heart and extreme intensification of the pulmonic second sound may suggest sclerosis of the pulmonary arteries but they may occur in the absence of that lesion if there is extensive fibrosis and destruction of pulmonary tissue. Gallop rhythm and premature auricular or ventricular contractions develop especially during febrile exacerbations or in the terminal stages of the tuberculous disease but fibrillation and block are unusual developments unless there is a complicating myocardial disease.

Arterial hypotension and low pulse pressure are the rule in phthisis but hypertension though encountered now and again is usually benign, perhaps due in part at least to a way of life relatively free from physical and psychic stress. The relatively low incidence of this latter syndrome may be due partly to the preponderance of active tuberculosis in youth and in early middle age.

Particularly bizarre circulatory changes may be noted if the heart and mediastinum are displaced as a result of pleuropulmonary infection. Symptoms are urgent often when pneumothorax or atelectasis occurs acutely, but these conditions can be recognized so readily that the source of the distress is apparent. However, if the mediastinal structures have been dislocated and normal anatomical relations have been distorted gradually, the signs may be confusing and diagnosis difficult. Traction of the heart to the right of the mid-line by fibrosis, adhesions, atelectasis or by massive excavation of the right lung may give rise to a variety of cardiac phonetics and may even lead to the thought of aneurysm of the arch of the aorta.

In short, somatic disturbances in the course of pulmonary tuberculosis may be so grouped as to resemble the circulatory imbalance of a host of extra-cardiac states or they may simulate closely organic disease of the heart itself, even when that organ is normal. Tuberculosis of the lungs may coexist with structural defects of the heart but this association leads relatively seldom to congestive failure of the myocardium.

The treatment is that of the underlying infectious process and if manifestations of secondary circulatory failure develop, those measures are indicated that are utilized whenever that condition occurs. Only in the event that residence in high altitudes is suggested or surgical treatment of the tuberculous focus is contemplated or that intensive antiluetic measures are suggested need the existence of organic cardiac disease influence seriously the choice of the therapeutic procedure.

CHARLES R. AUSTRIAN, M.D.,
Baltimore, Maryland.

SELECTED ABSTRACT

Korns, H. M. and Guinand, P. H.: Inequality of the Blood Pressure in the Brachial Arteries, with Especial Reference to Disease of the Arch of the Aorta. J. Clin. Inv. 12:143:1933.

Korns and Guinand confronted with several patients in whom the blood pressures were unequal in two arms without satisfactory explanation at autopsy, measured the blood pressure simultaneously in both arms of 1000 university students. They found an inequality of the pressures amounting to 10 mm. of mercury or more in 378 individuals. Nearly three-fourths of the higher pressures were in the right arm. There was no evidence that right or left handedness played any role in this regard. The higher pressure was not always in the same arm in a given individual on repeated examination. The authors concluded that inequality of the brachial artery pressures without organic disease is probably always transitory; that nearly all normal persons manifest it at one time or another; that it cannot be regarded as a sign of disease of the aorta or its branches unless it can be shown to be permanent. The physiology of these disparities in pressure is not understood.

M.N.F.